Journal of the American College of Cardiology
Vol. 49, No. 8, 2007

ISSN 0735-1097/07/$32.00

CORRESPONDENCE

Letters to the Editor

Stress Cardiomyopathy

We were interested to see the report of Hurst et al. (1) describing 4 women with stress (tako-tsubo) cardiomyopathy and systolic "midventricular ballooning." As noted by the investigators, this left ventricular (LV) contraction pattern differs from that of many other patients with stress cardiomyopathy because the distal portion of the chamber at the LV apex demonstrates a normal contraction pattern (apical sparing). Indeed, in our initial report of women with stress cardiomyopathy we also reported normal contraction of the apical LV segment in 9 of 22 patients (41%) based on cardiac magnetic resonance imaging (MRI) (2). In addition, Abdulla et al. (3) also recently reported apical sparing in 14 of 35 patients (40%) with stress cardiomyopathy. Therefore, this particular reversible pattern of abnormal LV contraction is very common in stress cardiomyopathy, and it may well have been an overestimation on the part of Hurst et al. (1) to regard this form of the condition as a novel variant.

Conversely, such patients clearly represent a subset within this disease spectrum, although of uncertain mechanism and clinical significance at this time. This diversity of phenotypic expression would, however, underscore the superiority of the term "stress cardiomyopathy" to describe this diverse entity rather than the ultimately confusing "midventricular ballooning" or "apical ballooning syndrome" (4–6). At this relatively early juncture in the evolving description of stress cardiomyopathy, application of clear and consistent nomenclature seems essential.

***Scott W. Sharkey, MD, FACC**
John R. Lesser, MD, FACC
Martin S. Maron, MD
Barry J. Maron, MD, FACC

*Minneapolis Heart Institute Foundation
920 East 28th Street, Suite 60
Minneapolis, Minnesota 55407
E-mail: scott.sharkey@allina.com

doi:10.1016/j.jacc.2006.12.004

REFERENCES

1. Hurst RT, Askew JW, Reuss CS, et al. Transient midventricular ballooning syndrome: a new variant. J Am Coll Cardiol 2006;48:579–83.
2. Sharkey SW, Lesser JR, Zenovich AG, et al. Acute and reversible cardiomyopathy provoked by stress in women from the United States. Circulation 2005;111:472–9.
3. Abdulla I, Kay S, Mussap C, et al. Apical sparing in tako-tsubo cardiomyopathy. Intern Med J 2006;36:414–8.
4. Bybee KA, Kara T, Prasad A, et al. Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. Ann Intern Med 2004;141:858–65.
5. Tsuchihashi K, Ueshima K, Uchida T, et al. Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction. Angina Pectoris-Myocardial Infarction Investigations in Japan. J Am Coll Cardiol 2001;38:11–8.
6. Park JH, Kang SJ, Song JK, et al. Left ventricular apical ballooning due to severe physical stress in patients admitted to the medical ICU. Chest 2005;128:296–302.

Reply

We appreciate the interest of Dr. Sharkey and colleagues in our report on transient midventricular ballooning of the left ventricle (1). Even after careful review of the study by Sharkey et al. (2), we are unable to find any evidence to substantiate their statement that "we also reported normal contraction of the apical LV [left ventricular] segment in 7 of 17 patients (41%) based on cardiac MRI [magnetic resonance imaging]." To the contrary, in their study the investigators state "*All* [our emphasis] exhibited a large wall-motion abnormality that involved akinesia or hypokinesia of the distal one-half to two-thirds of the LV chamber, which created a distinctive 'apical ballooning appearance'." Accordingly, we are unable to explain the discrepancy. The report by Abdulla et al. (3) was published after our study was submitted, making it impossible to have previously acknowledged.

Although the assertion that "this particular reversible pattern of abnormal LV contraction is very common in stress cardiomyopathy" may prove to be true, we did not comment on the prevalence of transient midventricular ballooning in our study. In fact, it would be anticipated that recognition of this midventricular variant would increase through a heightened awareness of transient ballooning syndrome, and this has proven correct as demonstrated in the report by Abdulla et al. (3), the recent MRI image from Steen et al. (4), and a case report by Shimizu et al. (5). We believe the "novel" aspect of the cases was recognizing the implications in determining the etiology underlying transient ballooning syndrome rather than the rarity of the occurrence.

The naming of this syndrome may be one of personal preference; however, we would suggest that "transient ballooning syndrome" as a descriptive nomenclature seems most appropriate. "Stress cardiomyopathy" implies a cause-and-effect relationship that, at present, has not been fully elucidated. Stress is ubiquitous, yet an associated transient cardiomyopathy is not!

***R. Todd Hurst, MD**
Christina S. Reuss, MD
A. Jamil Tajik, MD
J. Wells Askew, MD

*Mayo Clinic Arizona
Cardiovascular Diseases
13400 East Shea Boulevard
Desk 3A
Scottsdale, Arizona 85260
E-mail: hurst.todd@mayo.edu

doi:10.1016/j.jacc.2006.12.003